In the claims

1. (Currently amended) A pharmaceutical composition for increasing concentrations of chemokines to reduce entry of HIV virus into mononuclear cells through binding of chemokine binding receptors, the composition comprising at least one G1 phase arresting compound and at least one HIV viral entry inhibitor that inhibits entry of HIV to mononuclear cells, wherein the G1 phase arresting compound is in an amount sufficient to increase concentrations of extracellular β-chemokines, wherein the chemokines comprise MIP-1α, MIP-1β and RANTES.

2. Cancelled

3. (Previously presented) The pharmaceutical composition of claim 1, wherein the G1 phase arresting compound is a member selected from the group consisting of sodium butyrate, aphidicolin, hydroxyurea (HU), olomoucine, roscovitine, tocopherols, tocotrienols, and rapamycin (RAPA).

4. Cancelled

5. (Previously presented) The pharmaceutical composition of claim 1, wherein the HIV antiviral agent is a CCR5 inhibitors/antagonist, viral entry inhibitor or functional equivalent thereof.

6. (Previously presented) The pharmaceutical composition of claim 1, wherein the viral entry inhibitor is at least one member selected from the group consisting of: Fuzeon (T-20), SCH-C, SCH-D, PRO 140, TAK 779, TAK-220, AK602, UK-427, 857, NB-2, NB-64, T-649, T-1249, and functional analogs thereof that have the ability to inhibit entry of HIV into mononuclear cells.

7. (Previously presented) The pharmaceutical composition of claim 1, wherein the compound is administered orally, rectally, nasally, topically, vaginally or parenterally.

8. -9. Cancelled

10. (Currently amended) The pharmaceutical compositions of claim 1 ~~2~~, wherein the composition is administered in a cyclic therapy program.

11. (Withdrawn) A method for inducing increased levels of anti-HIV β-chemokines in activated lymphocytes, the method comprising:

administering a composition according to claim 1.

12. (Withdrawn) The method according to claim 11, wherein the G1 phase arresting agent is a member selected from the group consisting of: sodium butyrate, aphidicolin, hydroxyurea (HU), olomoucine, roscovitine, tocopherols, tocotrienols, and rapamycin (RAPA).

13.-14 (Cancelled

15. (Withdrawn) The method according to claim 11, wherein the HIV antiviral agent is CCR5 inhibitors/antagonist, viral entry inhibitor or functional equivalent thereof.

16. (Withdrawn) The method according to claim 1, wherein the viral entry inbititor is at least one member selected from the group consisting of: Fuzeon (T-20), SCH-C, SCH-D, PRO 140, TAK 779, TAK-220, AK602, UK-427, 857, NB-2, NB-64, T-649, T-1249, and functional analogs thereof that have the ability to inhibit entry of HIV into mononuclear cells.

17. (Withdrawn) The method according to claim 11, wherein the compound is administered orally, rectally, nasally, topically, vaginally or parenterally.

18. (Withdrawn and currently amended) A method for modifying synthesis of a receptor ligand to alter extracellular recognition of a receptor by an infectious agent, the method comprising:

administering to a cell at least one G1 phase arresting compound and at least one HIV viral entry inhibitor that inhibits entry of HIV to mononuclear cells, wherein the G1 phase arresting compound is in an amount sufficient to increase concentrations of extracellular β-chemokines and wherein the β-chemokines comprise MIP-1α, MIP-1β and RANTES and is the receptor ligand.

19.-22 (Cancelled)

23. (Withdrawn and currently amended) The method according to claim 18 ~~22~~, wherein G1 phase arresting compound is a member selected from the group consisting of: sodium butyrate, aphidicolin, hydroxyurea (HU), olomoucine, roscovitine, tocopherols, tocotrienols, and rapamycin (RAPA).

24. Cancelled

25. (Withdrawn) The method according to claim 18, wherein the viral entry inbititor is at least one member selected from the group consisting of: Fuzeon (T-20), SCH-C, SCH-D, PRO 140, TAK 779, TAK-220, AK602, UK-427, 857, NB-2, NB-64, T-649, T-1249, and functional analogs thereof that have the ability to inhibit entry of HIV into mononuclear cells.

26. (Withdrawn) The method according to claim 18, wherein the compound is administered orally, rectally, nasally, topically, vaginally or parenterally.

27. (Withdrawn and currently amended) A therapeutically effective method of combating a virus infection, the method comprising:

administering to a subject a therapeutically effective amount of a composition comprising a G1 phase arresting compound and at least one HIV viral entry inhibitor that inhibits entry of HIV to mononuclear cells, wherein the G1 phase arresting compound is in a concentration sufficient to induce increased levels and availability of β-chemokines thereby antagonizing the function of a chemokine receptor and reducing replication of the virus infection and wherein the β-chemokines comprise MIP-1α, MIP-1β or RANTES and the chemokine receptor is CCR5 .

28.-29. (Cancelled)

30. (Withdrawn) The method according to claim 27, wherein G1 phase arresting compound is a member selected from the group consisting of: sodium butyrate, aphidicolin, hydroxyurea (HU), olomoucine, roscovitine, tocopherols, tocotrienols, and rapamycin (RAPA).

31.-32. Cancelled

33 (Withdrawn) The method according to claim 30, wherein the viral entry inhibitor is at least one member selected from the group consisting of: Fuzeon (T-20), SCH-C, SCH-D, PRO 140,

TAK 779, TAK-220, AK602, UK-427, 857, NB-2, NB-64, T-649, T-1249, and functional analogs thereof that have the ability to inhibit entry of HIV into mononuclear cells.

34. Cancelled

35. (Withdrawn and currently amended) The method according to claim 30 ~~34~~, wherein the HIV vaccine and the G1 phase arresting agent are administered concurrently.

36. Cancelled..

37. (Withdrawn and currently amended) A method of maintaining viral control of an HIV infection, the method comprising:

administering at least one antiviral agent wherein the antiviral agent is at least one HIV viral entry inhibitor that inhibits entry of HIV to mononuclear cells in combination with at least one G1 phase arresting compound, wherein the G1 phase arresting compound is in a concentration sufficient to increase levels of β-chemokines and wherein the β-chemokines comprise MIP-1α, MIP-1β and RANTES.

38. (Withdrawn) The method according to claim 37, wherein the at least one antiviral agent and the at least one G1 phase arresting compound are administered concurrently.

39. The method according to claim 38, wherein the G1 phase arresting compound is a member selected from the group consisting of: sodium butyrate, aphidicolin, hydroxyurea (HU), olomoucine, roscovitine, tocopherols, tocotrienols, and rapamycin (RAPA).

40. (Withdrawn) The method according to claim 39, wherein the viral entry inbititor is at least one member selected from the group consisting of: Fuzeon (T-20), SCH-C, SCH-D, PRO 140, TAK 779, TAK-220, AK602, UK-427, 857, NB-2, NB-64, T-649, T-1249, and functional analogs thereof that have the ability to inhibit entry of HIV into mononuclear cells.

41. (Withdrawn) The method according to claim 40, wherein the G1 phase arresting agent is HU.

42. (Withdrawn) The method according to claim 41, wherein the G1 phase arresting agent is rapamycin.

43. (Withdrawn and currently amended) A therapeutically effective method to inhibit replication of HIV in a HIV infected subject, the method comprising:

a) administering at least one G1 phase arresting agent in a concentration sufficient to increase concentration of extracellular β-chemokines for a first predetermined time period, and wherein the β-chemokines comprise MIP-1α, MIP-1β and RANTES; and

b) administering the G1 phase agent with at least one antiviral agent, for a second predetermined time period, wherein the first and second time periods are sequential in a cyclic schedule.

44. (Withdrawn) The therapeutically effective method according to claim 43, wherein the G1 phase arresting agent is a member selected from the group consisting of: sodium butyrate, aphidicolin, hydroxyurea (HU), olomoucine, roscovitine, tocopherols, tocotrienols, and rapamycin (RAPA).

45. (Withdrawn) The therapeutically effective method according to claim 43, wherein the antiviral agent is a nucleoside RT inhibitor, CCR5 inhibitors/antagonist, viral entry inhibitor or functional equivalent thereof.

46. (Withdrawn) The therapeutically effective method according to claim 43, wherein the antiviral agent is at least one member selected from the group consisting of: Zidovudine (ZDV, AZT), Lamivudine (3TC), Stavudine (d4T), Didanosine (ddI), Zalcitabine (ddC), Abacavir (ABC), Emirivine (FTC), Tenofovir (TDF), Delaviradine (DLV), Efavirenz (EFV), Nevirapine (NVP), Fuzeon (T-20), Saquinavir (SQV), Ritonavir (RTV), Indinavir (IDV), Nelfinavir (NFV), Amprenavir (APV), Lopinavir (LPV), Atazanavir, Combivir (ZDV/3TC), Kaletra (RTV/LPV), Trizivir (ZDV/3TC/ABC), SCH-C, SCH-D, PRO 140, TAK 779, TAK-220, RANTES analogs, AK602, UK-427, 857, monoclonal antibodies, NB-2, NB-64, T-649, T-1249, and functional analog thereof.

47. (Withdrawn) The therapeutic method according to claim 43, wherein the cyclic schedule comprises:

a) administering a combination of at least one antiviral agent and at least one G1 phase arresting agent to the HIV infected subject for a predetermined first time period;

b) administering the at least one G1 phase arresting compound to the HIV infected subject for a second predetermined time period;

c) administering the combination of the antiviral agent and G1 phase arresting agent to the HIV infected subject for a predetermined third time period, which is less than the first period;

d) administering the G1 phase arresting compound to the HIV infected subject for a fourth predetermined time period which is less than the second time period; and

e) maintaining the cyclic schedule of steps c and d until replication of the HIV increases to a predetermined level.

48.-54 Cancelled